Review

Bruxism Is Unlikely to Cause Damage to the Periodontium: Findings From a Systematic Literature Assessment

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Background: This paper systematically reviews the MEDLINE and SCOPUS literature to answer the following question: Is there any evidence that bruxism may cause periodontal damage per se?

Methods: Clinical studies on humans, assessing the potential relationship between bruxism and periodontal lesions (i.e., decreased attachment level, bone loss, tooth mobility/migration, altered periodontal perception) were eligible. Methodologic shortcomings were identified by the adoption of the Critical Appraisal Skills Program quality assessment, mainly concerning the internal validity of findings according to an unspecific bruxism diagnosis.

Results: The six included articles covered a high variability of topics, without multiple papers on the same argument. Findings showed that the only effect of bruxism on periodontal structures was an increase in periodontal sensation, whereas a relationship with periodontal lesions was absent. Based on the analysis of Hill criteria, the validity of causation conclusions was limited, mainly owing to the absence of a longitudinal evaluation of the temporal relationship and dose-response effects between bruxism and periodontal lesions.

Conclusions: Despite the scarce quantity and quality of the literature that prevents sound conclusions on the causal link between bruxism and the periodontal problems assessed in this review, it seems reasonable to suggest that bruxism cannot cause periodontal damage per se. It is also important to emphasize, however, that because of methodologic problems, particularly regarding sleep bruxism assessment, more high-quality studies (e.g., randomized controlled trials) are needed to further clarify this issue. *J Periodontol 2015;86:546-555.*

KEY WORDS

Bruxism; periodontium; review.

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Bruxism is an umbrella term grouping different motor phenomena. Recently, an expert group reached consensus to define it as follows: "Bruxism is ... [a] repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.... Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism)."[1] Over the past few years, as part of an ongoing strategy to summarize the available findings on the argument, the potential clinical consequences of bruxism have been systematically reviewed, with focus on its effects on the temporomandibular joints and jaw muscles as well as on natural teeth and restored implant-supported dentitions.[2-4]

On the other hand, past theories suggested that bruxism also may be a potential risk factor for overload of the teeth-supporting tissues, i.e., the periodontium.[5,6] However, the literature on the topic has not yet been systematically reviewed. Nevertheless, over half a century, several studies on the periodontal effects of so-called occlusal trauma have been conducted.[7,8] Also, progressive modifications to the definition of occlusal trauma itself have been performed.[9] The most updated edition of the Glossary of Prosthodontic Terms[9] defined it as "trauma to the periodontium from functional or parafunctional forces causing damage to the attachment apparatus of

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the periodontium by exceeding its adaptive and reparative capacities." Hence, when occlusal forces exceed the adaptive capacity of the periodontal tissue, injury results. Within this framework, primary occlusal trauma is defined as the condition in which the pathologic occlusal forces are the main etiologic factor for changes in the periodontium, whereas secondary occlusal trauma occurs when the periodontium is already compromised by inflammation and bone loss.

In general, the more recent periodontal literature suggests that excessive forces on the dental occlusion are not likely to provoke any longstanding damage to a healthy periodontium, thus questioning the existence of a pure primary trauma.[10,11] Notwithstanding that, even though the presence of bacterial plaque as a cofactor seems to be considered a prerequisite for the onset of periodontal lesions in the presence of occlusal trauma as well, some authors have suggested that the literature is not conclusive.[12]

A possible explanation is that, given the variability of the potential clinical conditions leading to occlusal trauma, there is a need to determine the effects of each specific factor that may cause an excessive load on the teeth and their supporting structures. Based on this view, it must be remarked that most past investigations dealt mainly with the artificial insertion of an occlusal supra-contact, in either human or animal models, and the *ex juvantibus* effects of its removal in the presence or absence of facilitating cofactors such as bacterial plaque, fewer teeth, or reduced support.[13] In theory, trauma to the periodontium may also be due to excessive occlusal forces exerted during bruxism activities,[14] but it seems that such discrimination between trauma from bruxism in the natural environment and experimental trauma from artificial interferences has not yet been clearly demonstrated.

The aim of the present review is to provide an answer to the clinical research question "Is there any evidence that bruxism may cause periodontal damage per se?" by means of a systematic assessment of the available literature on the issue of bruxism-related effects on the periodontal structures.

MATERIALS AND METHODS

Search Strategy

On January 20, 2014, a systematic search in the medical literature was performed to identify all peer-reviewed English-language papers relevant to the review's aim. As a first step in the search strategy, the keyword term "bruxism" was used to browse the literature indexed in the two most qualified medical databases (National Library of Medicine's MEDLINE and SCOPUS) and retrieve lists of potentially relevant papers. Combinations of terms, including the words "clenching" or "grinding" alone and in association with the terms "periodontium," "periodontitis," "bone loss," "tooth/teeth mobility," or "bone resorption," were adopted. Based on title and abstract, the studies were selected for potential inclusion independently by two of the authors (DM and RM), who also performed data extraction and quality assessment by consensus. All authors contributed to the search expansion by checking for additional papers in the Google Scholar database, in the reference lists of potentially relevant papers, and in their own personal databases and institutional libraries.

The criteria for admittance in the systematic review were based on the type of study, and the inclusion was restricted to clinical studies on humans or animals that assessed the potential role of bruxism, as diagnosed with clinical assessment, questionnaires, interviews, polysomnography, or electromyography, as a causal factor for periodontal damage.

Systematic Assessment of Papers

The methodologic characteristics of the selected papers were assessed according to a format that enabled a structured summary of the articles in relation to four main issues: 1) patients/problem/population; 2) intervention; 3) comparison; and 4) outcome (PICO), for each of which specific questions were constructed.[15]

For each article, the study population (P) was described based on the criteria for inclusion, the demographic features of the sample, and the sample size. The study design was described in the section reserved to questions on the study intervention (I), and information was gathered on the approach to bruxism diagnosis. The comparison criterion (C) was based on the assessment of periodontium-related issues, by reporting the outcome variables, and the statistical approaches adopted by the authors to assess the role of bruxism as a risk factor for periodontal lesions. The study outcome (O) was evaluated in relation to the influence of bruxism on the presence of periodontal lesions.

Quality Assessment

In an attempt to increase the strength of this review, and in line with current needs to weigh the quality of the reviewed literature in systematic reviews, studies that were pertinent for inclusion underwent a quality assessment with the Critical Appraisal Skills Program (CASP) Cohort Study Checklist.[16] The CASP tool uses a systematic approach based on 12 specific criteria to appraise three broad areas: 1) study validity; 2) methodologic quality and presentation of results; and 3) external validity. The 12 criteria are: 1) Study issue is clearly focused. 2) Cohort is recruited in an acceptable way. 3) Exposure (bruxism) is

measured accurately. 4) Outcome (periodontal variables) is measured accurately. 5) Confounding factors are addressed. 6) Follow-up is long and complete. 7) Results are clear. 8) Results are precise. 9) Results are credible. 10) Results can be applied to the local population. 11) Results fit with available evidence. 12) There are important clinical implications. Each criterion can be given a response of yes, no, or "cannot tell," and each study can have a maximum score of 12. CASP scores were used to grade the methodologic quality of each study assessed.

Verification of Causality Criteria

The selected literature on the bruxism–periodontal damage relationship was also critically assessed in relation to this review's question: Is there any evidence that bruxism may cause periodontal damage per se?

To verify whether there is enough evidence for a cause-and-effect link between the two disorders, the widely adopted Hill criteria were used: 1) strength; 2) consistency; 3) specificity; 4) temporality; 5) gradient effect; 6) plausibility; 7) coherence; 8) experimental evidence; and 9) analogy.[17] Similar lists and modifications have often been used for discussing causation in the literature for bruxism, temporomandibular disorders, and dental occlusion.[18,19] Each paper was assigned one point for each criterion satisfied in favor of a positive bruxism–periodontal damage relationship, for a minimum score of 0 (no relationship between bruxism and periodontal damage) and a maximum of 9 points (absolute relationship between bruxism and periodontal damage).

RESULTS

The search identified 2,835 and 3,767 citations in the MEDLINE and SCOPUS databases, respectively, of which 2,562 were present in both databases. Thus, 4,040 citations were screened for eligibility. As shown in Figure 1, after excluding the citations that were clearly not pertinent for the review's aim on the basis of their title and abstract, nine papers were retrieved in full text and assessed for inclusion.[11,20-27] A consensus decision was made to exclude five of the nine papers. Reasons for exclusion were the following: 1) not dealing with bruxism in humans or animals (n = 2);[23,24] 2) adopting an unspecified strategy to provoke bruxism in monkeys (n = 1);[25] 3) presenting duplicated data of an included study (n = 1);[26] and 4) presenting a preliminary version of an included study (n = 1).[27] Thus, four papers were included in the review.[11,20,21] Search expansion strategies found two further papers,[28,29] accounting for a total of six papers included in the review.[11,20-22,28,29]

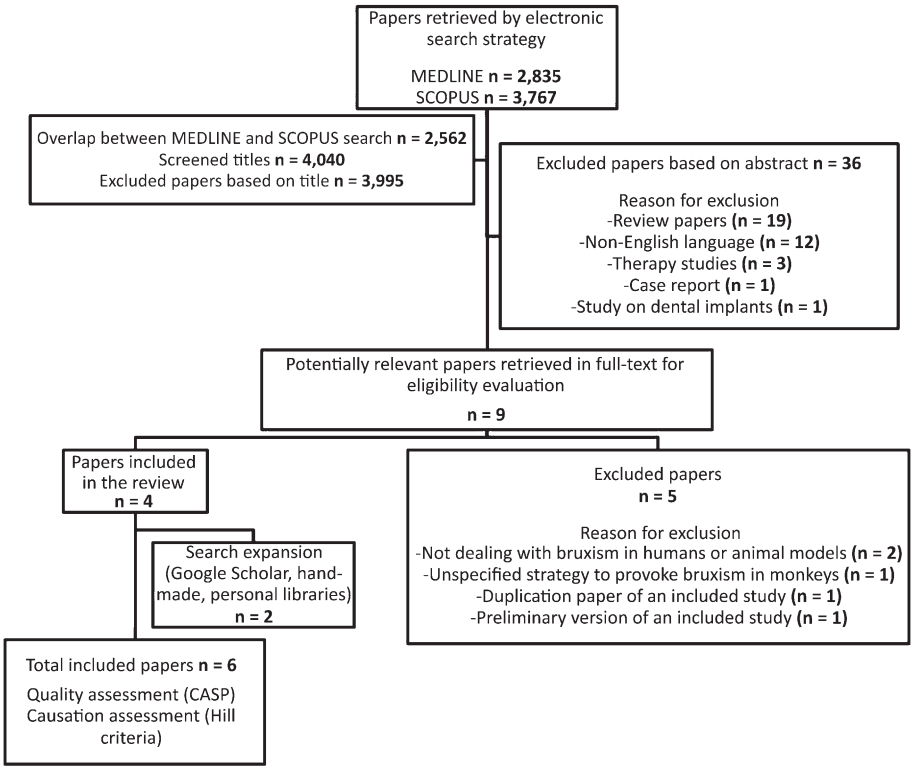

Figure 1.
Flowchart of the search strategy.

Structured reading of the included articles showed a high variability of topics. Two papers dealt with the influence of bruxism on periodontal perception by the assessment of interdental tactile threshold,[21,28] whereas single papers investigated the prevalence of periodontal problems in individuals with different grinding patterns,[20] the association between self-reported bruxism and periodontal problems at the general population level,[11] the prevalence of pathologic tooth migration in a cohort sample of periodontal patients in relation to self-reported bruxism,[29] and the differences in periodontal parameters between two cohorts of periodontal or bruxism patients.[22] Given the heterogeneity of study designs, meta-analysis of data could not be performed. Methodologic features and main findings concerning the possible relationship between bruxism and periodontal problems are summarized in

Table 1.

PICO Features of the Reviewed Studies

Reference	Population			Intervention	Comparison	Outcomes
	n (M/F)	Age range (years)	Characteristics			
Calderon et al. 2009[28]	115	14 to 37	Without severe TMD	• Case-control design (bruxers versus non-bruxers), unspecified size per group • Clinical bruxism diagnosis (tooth wear, shiny spots, masseter hypertrophy), three examiners	• MIT • Sensibility frequency to test with different-thickness foils • Multiple linear regression analysis (unclear strategy to manage data)	MIT 0.013 to 0.016 mm for both bruxers and non-bruxers; $P = 0.74$
Tokiwa et al. 2008[20]	50 (21/29)	Mean 41.2, 23 to 74	With stable occlusion	• Unspecified cohort-like design • Assessment of grinding types (canine versus molar grinding) • One-sided color foil	• AL • TM • NCL • DH • Measurement of variables in different types of bruxers	• Individuals with grinding patterns involving the molars have higher values of AL, TM, NCL, DH • No statistics for group comparison • No comparison versus non-bruxers
Ono et al. 2008[21]	28	Mean 26.3, 21 to 30	Students	• Case-control design (14 bruxers versus 14 non-bruxers) • Nocturnal masseter EMG	• Periodontal sensation by ITT • Tooth displacement • Mann–Whitney *U* test	• Mean ITT in bruxers lower than controls ($P < 0.001$) • Same pattern of force in voluntary clenching-induced tooth displacement, irrespective of bruxism status, but higher displacement in bruxers ($P < 0.05$)
Bernhardt et al. 2006[11]	2,980	20 to 79	Out of 4,310 individuals recruited for SHIP	• Cohort study • Self-reported bruxism (69% never; 23% sometimes; 8% often/always)	• PD • Clinical AL • Plaque score • Regression analysis	Bruxism not associated with PD or clinical AL
Martinez-Canut et al. 1997[29]	825 (297/528)	Mean 42.5, 19 to 72	Periodontal patients	• Cohort study • Self-reported bruxism (14%) or clenching (27%) • Attrition to confirm bruxism	• PTM as diagnosed on developing diastema in the maxillary anterior sextant • Wald coefficient	PTM: 15% of bruxers versus 12% of non-bruxers ($P = 0.16$); 26% of clenchers versus 28% non-clenchers ($P = 0.55$)

Table 1. (continued)
PICO Features of the Reviewed Studies

Reference	Population			Intervention	Comparison	Outcomes
	n (M/F)	Age range (years)	Characteristics			
Hanamura et al. 1987[22]	51 (26/25)	Mean 48.2, 35 to 60	Patients with moderate-to-severe periodontitis	Small, selected cohort study	BL	Higher BL in bruxers (88% of root length versus 72%; P <0.001)
	40 (19/21)	Mean 48.9, 37 to 62	Patients with bruxism/tooth wear	Self-reported bruxism diagnosis (57% in the bruxism group; 24% in the periodontitis group)	• AL • TM • Cross-tabulation statistics	• AL more pronounced in periodontal patients (P <0.001); AL >5 mm in 27.2% of periodontitis versus 4.9% of bruxer sites • TM more prevalent in periodontitis (P <0.001)

TMD = temporomandibular disorder; MIT = minimum interdental threshold; AL = attachment loss; TM = tooth mobility; NCL = non-carious cervical lesions; DH = dental hypersensitivity; EMG = electromyography; ITT = interocclusal tactile threshold; SHIP = Study of Health in Pomerania; PD = probing depth; PTM = pathologic tooth migration; BL = bone level.

Table 1. In general, the unique effect of bruxism on periodontal structures seems to be an increase in periodontal sensation.

Quality assessment showed that methodology was less than optimal, with only half of the investigations satisfying more than half of the quality items.[11,21,22] A common shortcoming of most studies was the self-reported approach to bruxism diagnosis, with the exception of a single paper providing electromyographic measurement of masseter muscle activity during sleep in the home environment.[21] Another point of major limitation was the unclear consistency of single papers' findings with respect to the available evidence, given the very poor literature on each specific topic. Thus, on average, the quality of investigations on the bruxism–periodontal lesions relationship can be improved and is currently not enough to provide high-quality evidence on the argument. Quality assessment of the individual papers is summarized in Table 2.

According to the analysis of Hill criteria,[17] whether a negative or positive causal relationship between bruxism and periodontal lesions was claimed by the authors of the individual papers, the conclusions on causation satisfied at least half of the criteria in only three papers.[11,21,22] Common shortcomings to all papers were the absence of any information about the temporal relationship and on the gradient effect, i.e., dose-response effect, due to the lack of any longitudinal observations. Also, given the paucity of literature on the topic, very little information could be retrieved as far as consistency, coherence, and analogy criteria are concerned. Assessment of validity of causation conclusions for individual papers is summarized in Table 3.

DISCUSSION

For decades, debate on the role of trauma from dental occlusion in the etiology of periodontal disease has attracted generations of researchers and dental practitioners.[7,8,30] Despite a general tendency to agree that occlusal factors alone cannot explain the onset of periodontal disease, which is instead inflammatory/infective in nature, the argument still animates discussions and is still worthy to be summarized in more recent reviews.[10,31,32] Within the factors that may exert forces on the periodontium, bruxism might be hypothesized to be a possible cause of overload. Thus, in line with recent papers that summarized several aspects of the potential pathologic consequences of bruxism,[2,3,33] the present review aims to provide a summary of the literature on the effects of bruxism on the periodontium. Unfortunately, the review falls short in the attempt to provide sound conclusions because of shortcomings in the literature.

Table 2.

CASP Quality Assessment of the Reviewed Papers

Reference	Item 1	Item 2	Item 3	Item 4	Item 5	Item 6	Item 7	Item 8	Item 9	Item 10	Item 11	Item 12	Total Quality Score (0 to 12)
Calderon et al. 2009[28]	Yes	No (unclear recruitment strategy)	Yes (even if clinical, not measurement-based, diagnosis)	No (MIT measured with foils)	No (no evaluation of teeth-related factors)	No (experiment without multiple observation points)	Yes	No (outcome variable not measured accurately)	No (outcome variable not measured accurately)	Yes	Cannot tell (other studies not available)	No (unclear implications)	4
Tokiwa et al. 2008[20]	Yes	Yes	No (subjective strategy to assess grinding patterns; no calibrated examiners)	Yes	No (other causes of periodontal problems not assessed)	No (experiment without multiple observation points)	No (conclusions not clearly stated)	No (no statistical analysis)	No (no statistical analysis)	Yes	Cannot tell (other studies not available)	No (unclear implications)	4
Ono et al. 2008[21]	Yes	Yes	Yes	Yes	Yes	No (experiment without multiple observation points)	Yes	Yes	Yes	Yes	Cannot tell (other studies not available)	Yes	10
Bernhardt et al. 2006[11]	Yes	Yes	No (self-reported bruxism)	Yes	Yes	No (cross-sectional)	Yes	Yes	Yes	Yes	Yes	Yes	10
Martinez-Canut et al. 1997[29]	Yes	Yes	No (self-reported bruxism)	No (unspecified periodontal problems and unvalidated PTM diagnosis)	Yes	No (cross-sectional)	Yes	No (unvalidated diagnoses)	Yes	Yes	Cannot tell (other studies not available)	No (unclear implications)	6
Hanamura et al. 1987[22]	Yes	Yes	No (self-reported bruxism)	Yes	Yes	No (cross-sectional)	Yes	Yes	Yes	Yes	Cannot tell (other studies not available)	Yes	9

MIT = minimum interdental threshold; PTM = pathologic tooth migration.

Table 3.

Assessment of Validity of Causation Conclusions of the Reviewed Studies According to Hill Criteria

Reference	Main Causation Finding	Item 1	Item 2	Item 3	Item 4	Item 5	Item 6	Item 7	Item 8	Item 9	Total Score for Validity of Causation Conclusion
Calderon et al. 2009[28]	Bruxism does not alter minimum interdental threshold	No (lack of internal validity)	NA	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	NA	No (not in line with other experiments)	No (different findings with respect to Ono et al.[21])	2
Tokiwa et al. 2008[20]	Grinding patterns involving the molar are associated with more periodontal problems	Yes	NA	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	NA	Yes (even if only experiment on the issue)	NA	4
Ono et al. 2008[21]	Bruxism reduces interocclusal tactile threshold	Yes	Yes	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	NA	Yes (even if findings are different from the study of Calderon et al.[28])	Yes	5
Bernhardt et al. 2006[11]	Bruxism is not associated with reduced CAL or increased PD	Yes	Yes	No (study of bruxism-periodontitis relationship was not the primary aim)	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	Yes	No (no experimental designs on the issue)	Yes	5
Martinez-Canut et al. 1997[29]	Bruxism is not associated with pathologic tooth migration	Yes	NA	No (unclear outcome variable)	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	NA	No (no experimental designs on the issue)	Yes	3
Hanamura et al. 1987[22]	Bruxers have fewer periodontal problems than periodontal patients	Yes	NA	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	Yes	No (no experimental designs on the issue)	Yes	5

NA = not applicable, CAL = clinical attachment level, PD = probing depth.

First, it should be noted that very few research papers on the argument were published, with only six papers admitted in the review.[11,20-22,28,29] Such limited quantity causes serious concerns about the external validity of each individual paper.[34] The strategies adopted to assess the consequences of bruxism on the periodontium varied widely and prevented any attempts to meta-analyze the data. Moreover, the specificity of bruxism diagnosis was, on average, poor, since it was based on a self-reported approach as the only diagnostic method in almost all papers. Unfortunately, such an approach is suitable to detect only possible bruxism at best[1] and causes serious concerns about the internal validity of an individual investigation on any bruxism issue.[35]

Second, the quality of the reviewed literature was, on average, low. The failure to provide a validated bruxism diagnosis, the absence of multiple observation points, and the lack of multiple papers on the same topic (and thus inability to control papers for consistency with the available evidence) are the main shortcomings identified with quality assessment. In particular, even two of the three highest-quality papers did not use the standard of reference diagnostic approach to bruxism diagnosis.[11,22] Such a limitation should have been prevented by selecting only those papers adopting bruxism measurement diagnosis, thus potentially avoiding any reviewer bias in quality assessment. On the other hand, other factors—such as the very low number of polysomnography-based papers in the whole bruxism literature[2,3,33] and the usefulness of polysomnography itself to detect clinically meaningful bruxism currently being under validity appraisal[4]—suggested that papers in this review be included independently of the diagnostic approach to bruxism.

Third, mainly as a consequence of the above, the validity of causation conclusions was, in general, limited. Again, the absence of an evaluation on the temporal relationship and dose-response effects between bruxism and periodontal lesions, as well as the poor specificity with respect to the study aims concerning bruxism, were identified as the critical factors that prevented the definitive confirmation or refutation of a causal link between the two conditions.

In theory, the possibility that some part of the historical literature dealing with the generic topic of occlusal trauma should have actually focused on trauma from bruxism or parafunctions, thus being potentially worthy of inclusion in the review, cannot be ruled out. On the other hand, in practice, such a possibility is unlikely, and the review's findings may have been influenced, given the very poor specificity for a bruxism diagnosis even in the reviewed papers themselves. The choice of excluding papers was particularly difficult in the case of some animal studies.[23,25] Indeed, among the several investigations on the role of occlusal trauma in animal models, some claimed to assess the effects of bruxism according to the purported bruxism-provoking effects of artificially inserted high occlusal restorations.[23,25] In contrast, human studies have dismantled the role of natural malocclusion and/or artificially high restorations as causal factors for bruxism.[36,37] Thus, such animal studies were excluded from the review because of their different a priori speculations with respect to the current knowledge on humans. In any case, it should be pointed out that the study authors claimed the absence of any clinical or histologic evidence that bruxism had caused a progression of gingivitis to destructive, chronic marginal periodontitis in monkeys.[23,25] Such findings are in line with the experiences of artificially created occlusal trauma in dogs,[38] thus supporting the concept that, whatever the origin of trauma on the periodontium (i.e., bruxism or occlusion) or the model under study (i.e., human or animal), excessive forces cannot be viewed as the sole factor that determines periodontal damage.

Taking the above factors into consideration, findings from the reviewed literature seem to suggest the absence of a causal link between bruxism and periodontal damage. It seems reasonable to hypothesize that an increased periodontal perception is the only plausible bruxism consequence on the periodontium.[21] The absence of any radiologic investigation makes it impossible to hypothesize whether such increased perception is due to an enlargement of the periodontal ligament or any other factors. However, the issue has important clinical implications, especially with respect to the need for adopting prudent prosthodontics strategies in bruxers.[39]

Although it may be suggested that bruxism cannot cause periodontal damage per se, several clinical questions remain unanswered due to the very poor quantity and less-than-optimal quality of the papers included in this review. For instance, an interesting topic for future investigations might be the assessment of bruxism prevalence in periodontal patients (the other side of the coin). Indeed, from a theoretic viewpoint, it is also possible that teeth with decreased periodontal support or a certain degree of mobility may act as a protective mechanism against bruxism via the same pathways that reduce bruxism activities in individuals with high restorations. The role of bruxism itself as a real source of trauma to the periodontium should be carefully appraised, especially in the light of increasing evidence that several different motor activities with potentially different etiologies are actually grouped under the umbrella term "bruxism." Thus, the effects of such trauma on the periodontium, if existing, should be studied separately

based on the possible consequences of teeth clenching or grinding. Also, the possibility has to be addressed that bruxism, even if not a cause of pure primary trauma, may precipitate conditions and jeopardize survival of migrated or periodontally migrated teeth. Finally, as a strong recommendation for the future, it is of paramount importance that homogeneous definitions and strategies to diagnose bruxism as well as multidisciplinary approaches (i.e., periodontists and bruxism/orofacial pain experts) are adopted.

CONCLUSIONS

This paper systematically reviewed the available bruxism literature to answer the following question: Is there any evidence that bruxism may cause periodontal damage per se? Unfortunately, the scarce quantity and quality of the reviewed literature prevented sound conclusions. Despite no positive relationship being found between bruxism and the periodontal problems assessed in this review, possibly suggesting that bruxism cannot cause periodontal damage per se, it is also important to emphasize that the methodologic problems, regarding particularly sleep bruxism assessment, require more high-quality studies (e.g., randomized controlled trials) to be performed to further clarify this issue.

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